CLAIMS:

The applicant claims the following:

1. A compound consisting of a hydrazide molecule as a part of its construction, where the carboxylic acid moiety of said hydrazide molecule is provided by any organic acid, and where the hydrazine moiety of said hydrazide molecule is provided by hydrazine.

2. The compound according to claim 1, where said hydrazine moiety has a substituent other than hydrogen.

3. The compound according to claim 1, where said carboxylic acid moiety is provided by nicotinic acid and where said hydrazine moiety is provided by isopropyl hydrazine.

4. The compound according to claim 1, to provide pharmaceutical use as a protease inhibitor drug that shuts down disease activated protease enzymes.

5. A method of treatment for a condition, disorder, or disease by administering to a patient in need thereof an effective amount of a pharmaceutical compound according to claim 1.

6. The method according to claim 5 where said disease is a viral infection which includes but not limited to a member belonging to arenaviridae, bunyaviridae, caliciviridae, coronaviridae, flaviviridae, hepadnaviridae, herpesviridae, orthomyxoviridae, papovaviridae, paramyxoviridae, parvoviridae, picornaviridae, poxviridae, reoviridae, retroviridae, rhabdoviridae, and togaviridae families that includes members as adenovirus, hepatitis type A, hepatitis C, hepatitis B, herpes simplex type I, herpes simplex type II, human T-lymphotropic retrovirus III, human immunodeficiency virus, influenza, influenza, measles virus, mumps virus, papilloma virus, papova virus, rhinovirus, rubella virus, SARS crown virus and varicella whereby said dysfunctional shutdown of protease provides a cessation of peptides and viral coat protein needed for replication and is inductive of cell maintenance disease eradication processes.

7. The method according to claim 5 where said disease is a prion infection which includes but not limited to Creutzfeldt-Jakob Disease, Gerstmann-Straussler-Scheinker Disease, kuru, and bovine spongiform encephalopathy whereby said dysfunctional shutdown of protease provides a cessation of prion

protein production and is inductive of cell maintenance disease eradication processes.

8. The method according to claim 5 where said disease is cancer which includes but not limited to acoustic neuroma, acute granulocytic leukemia, adenocarcinoma, adrenal cortex carcinoma, angiosarcoma, astrocytoma, basal cell carcinoma, bile duct cancer, bladder carcinoma, breast cancer, bronchogenic carcinoma, cervical endometrial carcinoma, cervical carcinoma, cervical hyperplasia, chordoma, choriocarcinoma, chronic sarcoma, colon carcinoma, craniopharyngioma, cystadenocarcinoma, endotheliosarcoma, ependymoma, epithelial carcinoma, esophageal carcinoma, thrombocytosis, fibrosarcoma, glioma, hairy cell pancreatic cancer, neck carcinoma, hemangioblastoma, hepatoma, Kaposi's sarcoma, leiomyosarcoma, leukemia, liposarcoma, lung carcinoma, Hodgkin's disease, macroglobulinemia, malignant hypercalcemia, carcinoid carcinoma, pancreatic insulinoma, malignant melanoma, medullary carcinoma, medulloblastoma, melanoma, meningioma, multiple myeloma, mycosis fungoides, myxosarcoma, neuroblastoma, oligodendroglioma, genitourinary carcinoma, osteogenic sarcoma, ovarian carcinoma, pancreatic carcinoma, papillary adenocarcinomas, pinealoma, polycythemia mesothelioma, primary macroglobulinemia, primary brain carcinoma, prostatic carcinoma, renal cell carcinoma, retinoblastoma, leukemias, rhabdomyosarcoma, sarcoma, sebaceous gland carcinoma, seminoma, skin cancer, small cell lung carcinoma, soft-tissue lymphangiosarcoma, squamous cell carcinoma, stomach granulocytic leukemia, sweat vera, synovioma, testicular tumor, testicular carcinoma, and thyroid carcinoma whereby said dysfunctional shutdown of protease halts protein and peptide production necessary for cancerous cell division, growth, and metastasis, and is inductive of cell maintenance disease eradication processes.

9. The method according to claim 5 where said condition is caused by protease production of toxic or aberrant proteins that includes but not limited to multiple myeloma, Alzheimer's disease, Parkinson's disease, and other disorders whereby said dysfunctional shutdown of protease halts toxic protein production and is inductive of cell maintenance action to restore the peptide genetic encoding sequence or to terminate such cell.

10. The method according to claim 5 where said disorder is a loss of systemic control or biological regulation processes caused by slight alterations in proteins, peptides, hormones, or prostaglandin products as used to communicate or control biological regulation processes manifested as conditions that includes but not limited to acne vulgaris, adenomatous intestinal polyposis, adenosine deaminase deficiency, alcoholism and other addictions, anemia, angina pectoris, anorexia, arteriosclerosis, carbonic anhydrase deficiency syndrome, classical phenylketonuria, colitis, collagenous diseases, Crohn's disease, cystic

fibrosis, diabetes mellitus, Gaucher's disease, glaucoma, glucose-6-phosphste dehydrogenase deficiency syndrome, high cholesterol, hypercholesterolemia, hypersarkosinemia, hypertension, spontaneous tumors, malignant melanoma, multiple sclerosis, muscular dystrophy, obesity, osteopetrosis, psoriasis, schizophrenia, severe or neurotic pain, spondylitis, t-cell immunodeficiency, triosephosphate isomerase deficiency syndrome, and ulcerative colitis whereby said dysfunctional shutdown of protease is inductive of cell maintenance action to restore peptide sequence integrity responsible for such systemic communications breakdown.

11. The method according to claim 5 wherein said condition is a microorganism infection which includes infections caused by organisms belonging to absidia, acanthamoeba, acanthocephala, aspergillus, balantidium, basidiomycota, blastomyces, candida, cestoda, cestodes, chaetognatha, cladosporium, coccidioides, comycota, cryptococcus, cycliophora, deutoeromycota, entamoeba, gastrotricha, geotrichum, histoplasma, leishmania, leptomyxida, mucor, naegleria, nematoda, paracoccidioides, phialophora, plasmodium, platyhelminthes, pneumocystis, pseudoallescheria, rhizopus, rhodotorula, rotifera, sporothrix, torulopsis, toxoplasma, trematoda; aschelminthes, trematodes, trypanosoma, xylohypha, and zygomycota whereby said dysfunctional shutdown of protease halts peptide production necessary for cell division, reproduction, and proliferation of said organisms.

12. The method according to claim 5 where said disease is a bacterial infection caused by any member of the species related to actinomycosis, anthrax, bartonellosis, borreliosis, brill-zinsser, brucellosis, campylobacter, chlamyldial, cholera, clostridial bacteroides, ehrlichiosis, enterobacteriaceae, erysipelothricosis, gonorrhea, hemophilus, legionella, leptospirosis, listerosis, melioidosis, mycoplasma, neisseria, nisseria, nocardiosis, noncholera vibrio, plague, pneumoccal, pseudomonas, rickettsioses, salmonella,
shigellosis, spirochetes, staphyloccal, steptoccal, streptbacillus, syphilis, treponematoses, and tulamremia whereby said dysfunctional shutdown of protease halts peptide production necessary for cell division, reproduction, and proliferation of any member of said species.

13. The method according to claim 5 wherein said condition is a need to prevent antibiotic resistant strains of organisms from evolving as a result of antibiotic use whereby said dysfunctional shutdown of protease that is innate to said organisms halts peptide production necessary for cell division, reproduction, and proliferation of progeny that could pass on antibiotic or drug resistant traits to successive generations.

14. The method according to claim 5 wherein said condition is a need to prevent and infection from worsening while testing antibiotic of unknown efficacy whereby said dysfunctional shutdown of protease that is innate to said organisms halts peptide production necessary for cell division, reproduction, and proliferation.

15. The method according to claim 5 to prevent disease or illness from occurring by providing prophylaxis treatment whereby such use serves to halt disease action before it can establish sufficiently to cause systemic affects or damage.

16. The method according to claim 5 where said condition is systemic induced cell death or apoptosis, caused by triggering of innate genetic programs existing in the chromosomes of cells caused by injury, age, or stresses, and as can result from hypoxia or ischemia, whereby said dysfunctional shutdown of protease provides a cessation of peptide signals necessary to induce apoptotic destruction of endangered cells.

17. The method according to claim 5 where said disorder is obesity or weight control whereby said dysfunctional shutdown of protease provides a cessation of peptides that induce fat cell division thereby causing the excretion of lipids that cannot be stored in the existing population of fat cells.

18. The method according to claim 5 where said disorder is an inability for systemic biological regulation processes to communicate feelings of satisfaction or hunger based on biological needs such that abusive behavior as over eating, over indulgence, or addictive behavior results, whereby said dysfunctional shutdown of protease is inductive of cell maintenance action to restore peptide sequence integrity responsible for such systemic communications breakdowns.

19. A process discovered as provided by a protease enzyme reaction to a hydrazide substrate
consistent with hydrazide substrate targeting by protease enzyme cleavage action,
where said cleavage action releases an active hydrazine radical,
where said hydrazine radical attaches irreversibly to the protease enzyme,
where said protease enzyme is rendered dysfunctional and inactive,
where said protease dysfunction halts the production of disease associated proteins,
where said dysfunctional state induces cell maintenance action,
where said cell maintenance action restores healthy cell operation,
where a disease free condition is thereby provided.

20. A method for providing a hydrazide substrate, protease enzyme inhibitor type therapeutic action by using any one of the existing hydrazide substrate, oxidase enzyme inhibitor type drugs which have identical therapeutic action exemplified by iproniazid, isocarboxazid, and nialimide which have a 50 year history of safe medical use.